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L2: Entry 1 of 1 File: JPAB Jun 13, 1995

PUB-NO: JP407149786A
DOCUMENT-IDENTIFIER: JP 07149786 A
TITLE: GLYCEROGLYCOLIPID AND CARCINOGENIC PROMOTER INHIBITOR

PUBN-DATE: June 13, 1995

INVENTOR-INFORMATION:
NAME
YAZAWA, KAZUYOSHI
SAKAKIBARA, NISAKU
WATANABE, MIYAKO
NAGATSU, AKITO
TOKUDA, HARUKUNI

ASSIGNEE-INFORMATION:
NAME COUNTRY
SAGAMI CHEM RES CENTER N/A

APPL-NO: JP05319188
APPL-DATE: November 26, 1993

INT-CL (IPC): C07H 15/06; A61K 31/70

ABSTRACT:

PURPOSE: To obtain a new glyceroglycolipid having strong carcinogenic promoter inhibiting action and low cytotoxicity and useful as a carcinogenic promoter inhibitor effective as an active component of a cancer preventing or treating agent, etc.

CONSTITUTION: New glyceroglycolipid is expressed by formula I (R is H or a hydroxyl-protecting group; R1 and R2 are an acyl residue of a 12-24C fatty acid provided that at least one of R1 and R2 is eicosapentaenoyl or docosahexaenoyl) (e.g. 1-O-eicosapentaenoyl-2-O-myristoyl-3-O-β-D-galacropyranosyl-sn-glycerol), and has strong carcinogenic promoter inhibiting action and low cytotoxicity, and is effective as a cancer preventing or treating agent. The compound can be produced by reacting a brominated α-D-galactopyranosyl of formula II (Ac is acetyl) with 1, 2-di-O-benzylglycerol of formula III (Bn is benzyl) and subjecting the reaction product to the deprotection of hydroxyl group, protection and acylation.

L7 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
ACCESSION NUMBER: 1996:201771 CAPLUS
DOCUMENT NUMBER: 124:256340
TITLE: Signal transduction through lipid second messengers
AUTHOR(S): Spiegel, Sarah; Foster, David; Kolesnick, Richard
CORPORATE SOURCE: Dep. Biochem. and Mol. Biol., Georgetown Univ. Med. Center, Washington, DC, 20007, USA
SOURCE: Current Opinion in Cell Biology (1996), 8(2), 159-67
CODEN: COCBE3; ISSN: 0955-0674
PUBLISHER: Current Biology
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 82 refs. This review emphasizes the generation of **glycerolipid** and sphingolipid second messengers, and their mol. targets. The role of the phosphatidylinositol transfer protein and phospholipase D in signal transmission, and the structures of the 1,2-diacylglycerol transfer protein and phospholipase D in signal transmission, and the structures of the 1,2-diacylglycerol and calcium-binding sites of protein kinase C are discussed. Further, ceramide signaling through protein kinases and the role of cross-talk in the signaling of **apoptosis** and inflammation are addressed.

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L1: Entry 1 of 1 File: JPAB Jan 31, 1985

PUB-NO: JP360019716A
DOCUMENT-IDENTIFIER: JP 60019716 A
TITLE: ANTITUMOR AGENT

PUBN-DATE: January 31, 1985

INVENTOR-INFORMATION:
NAME
NOJIMA, SHOSHICHI
NOMURA, MASAAKI

ASSIGNEE-INFORMATION:

NAME	COUNTRY
TAKEDA CHEM IND LTD	N/A
NOJIMA SHOSHICHI	N/A

APPL-NO: JP58126437
APPL-DATE: July 11, 1983

US-CL-CURRENT: 514/23; 514/53
INT-CL (IPC): A61K 31/70; C07H 15/08

ABSTRACT:

PURPOSE: An antitumor agent effective for remedying warm-blooded animals seized with a malignant tumor such as leukemia, solid cancer, etc., containing glyceroglycolipid.

CONSTITUTION: An antitumor agent containing a glyceroglycolipid shown by the formula (R1 and R2 are 8∼30C aliphatic hydrocarbon residue; R3 is glycosyl of monosaccharide or disaccharide). It can be safely administered orally (e.g., tablet, granule, powder, capsule, syrup, emulsion, suspension, etc.) or parenterally (e.g., injection, suppository, etc.). A dose is 0.1∼100mg/kg/day, preferably 0.5∼ 30mg/kg/day, and applied daily or 2∼7 days interval. It may be administered 1∼3 times dividedly a day.

L6 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:338132 CAPLUS
DOCUMENT NUMBER: 128:326478
TITLE: **Apoptosis** inducers from animals, plants or microorganisms
INVENTOR(S): Sakai, Takeshi; Koyama, Nobuto; Tatsumi, Yoko; Sagawa, Hiroaki; Yu, Fu-Gong; Ikai, Katsushige; Kato, Ikunoshin
PATENT ASSIGNEE(S): Takara Shuzo Co., Ltd., Japan; Sakai, Takeshi; Koyama, Nobuto; Tatsumi, Yoko; Sagawa, Hiroaki; Yu, Fu-Gong; Ikai, Katsushige; Kato, Ikunoshin
SOURCE: PCT Int. Appl., 71 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
INT. PATENT CLASSIF.:
MAIN: A61K035-78
SECONDARY: A61K035-80; A61K035-84; A61K035-66; A61K038-00
CLASSIFICATION: 63-4 (Pharmaceuticals)
Section cross-reference(s): 1, 10, 11, 12
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9820884	A1	19980522	WO 1997-JP3997	19971031
W: AU, BR, CA, CN, JP, KR, MX, US, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9747269	A1	19980603	AU 1997-47269	19971031
EP 941737	A1	19990915	EP 1997-909728	19971031
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
CN 1233961	A	19991103	CN 1997-198922	19971031
PRIORITY APPLN. INFO.:			JP 1996-311224	19961108
			JP 1996-356416	19961226
			WO 1997-JP3997	19971031

ABSTRACT:
The invention relates to **apoptosis** inducers or carcinostatic agents characterized by contg. as the active ingredient glycerolipids and/or glyceroglycolipids from animals, plants or microorganisms.

SUPPL. TERM: **apoptosis** inducer **glycerolipid** glyceroglycolipid; carcinostatic agent glycerolipids glyceroglycolipid
INDEX TERM: Algae
Animal
Antitumor agents
Eggplant (Solanum melongena)
Fermentation
Microorganism
Mushroom
Plant (Embryophyta)
Rice bran
Seaweed

Spinach (Spinacia oleracea)
Tea products
(**apoptosis** inducers from animals, plants or microorganisms)

INDEX TERM: Glycerolipids
ROLE: PUR (Purification or recovery); THU (Therapeutic use);
BIOL (Biological study); PREP (Preparation); USES (Uses)
(**apoptosis** inducers from animals, plants or microorganisms)

INDEX TERM: Glycerolipids
Glycolipids
ROLE: PUR (Purification or recovery); THU (Therapeutic use);
BIOL (Biological study); PREP (Preparation); USES (Uses)
(glyceroglycolipids; **apoptosis** inducers from animals, plants or microorganisms)

INDEX TERM: **Apoptosis**
(inducers; **apoptosis** inducers from animals,

L12 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1980:74554 CAPLUS
DOCUMENT NUMBER: 92:74554
TITLE: **Lipid** degradation during manufacture of black **tea**
AUTHOR(S): Wright, Anthony J.; Fishwick, Michael J.
CORPORATE SOURCE: ARC Food Res. Inst., Norwich, NR4 7UA, Engl.
SOURCE: Phytochemistry (1979), 18(9), 1511-13
CODEN: PYTCAS; ISSN: 0031-9422
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Approx. 85% of the fatty acids liberated during the manuf. of black **tea** was due to autolysis of phosphatidylcholine, monogalactosyldiglyceride, digalactosyldiglyceride, and phosphatidylethanolamine in **tea** leaf tissue. Linolenic acid [463-40-1], linoleic acid [60-33-3], and palmitic acid [57-10-3] were the principal fatty acids released from these **lipids**, accounting for .apprx.90% of the fatty acids released, and all 3 underwent further degrdn. Linoleate (60% of the fatty acids released) was mainly derived from galactolipids, and thus the upper limit of release depended upon chloroplast maturity and content of the leaf tissues. **Lipid** breakdown was complete after 2 h fermn., and, as there was apparently no accumulation of long chain fatty acid intermediates, volatile prodn.

L22 ANSWER 5 OF 5 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1996:370613 BIOSIS
DOCUMENT NUMBER: PREV199699092969
TITLE: Effects of nonsteroidal anti-**inflammatory** drugs on proliferation and on induction of **apoptosis** in colon cancer cells by a prostanglandin-independent pathway.
AUTHOR(S): Hanif, Rashid; Pittas, Anastasios; Feng, Yan; Koutsos, Markos I.; Qiao, Liang; Staiano-Coico, Lisa; Shiff, Steven I.; Rigas, Basil (1)
CORPORATE SOURCE: (1) Dep. Med. F-231, N. Y. Hosp.-Cornell Med. Cent., 525 E. 68th St., New York, NY 10021 USA
SOURCE: Biochemical Pharmacology, (1996) Vol. 52, No. 2, pp. 237-245.
ISSN: 0006-2952.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Nonsteroidal anti-**inflammatory** drugs (NSAIDs) decrease the incidence of and mortality from colon cancer. We observed that NSAIDs inhibit the proliferation rate, alter the cell cycle distribution, and induce apoptosis in colon cancer cell lines. We evaluated whether the inhibition by NSAIDs of prostaglandin (PG) synthesis is required for their effects on colon cancer cells by studying two human colon cancer cell lines: HCT-15 and HT-29. HCT-15, which lacks cyclooxygenase transcripts, does not produce PGs even when exogenously stimulated, whereas HT-29 produces PGE-2, PGF-2alpha, and PGI-2. HCT-15 and HT-29 cells, when treated for up to 72 hr with 200 mu-M sulindac sulfide (an active metabolite of sulindac) or 900 mu-M piroxicam, showed changes in proliferation, cell cycle phase distribution, and apoptosis. Treatment with PGE-2, PGF-2alpha, and PGI-2, following a variety of protocols, and at concentrations between 10-6 and 10-11 M, failed to reverse the effects of NSAIDs on these three parameters of cell growth. We concluded that NSAIDs inhibit the proliferation rate of the two colon cancer cell lines independent of their ability to inhibit PG synthesis. Thus, alternative mechanisms for their activity on tumor cell growth must be entertained. These observations may be relevant to the mechanism of colon tumor inhibition by NSAIDs.